Effect of Whiskey on Atrial Vulnerability and "Holiday Heart"

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Vulnerability to atrial fibrillation and flutter was examined in 11 alcohol abusers who did not have cardiomyopathy or manifest heart failure. Atrial extrastimulation was done with rapid pacing (drive cycle length 500 ms) to facilitate induction of atrial vulnerability, seen in four alcohol abusers. The remaining seven were retested 30 minutes after drinking 60 to 120 ml of 86 proof whiskey (ethanol blood levels were 49 to 101 mg/100 ml but pulmonary capillary wedge pressure remained normal in all) and atrial fibrillation or flutter was induced in three of the drinkers. Three nondrinkers, symptomatic with sinus bradycardia but not in heart failure, were found not to be vulnerable to atrial fibrillation or flutter, but flutter was induced in two of the three after drinking whiskey. Whiskey did not alter atrial functional refractory periods (mean ± standard error of the mean 297 ± 14 to 290 ± 12 ms) or widen the dispersion among three disparate right atrial sites (57 ± 13 to 47 ± 12 ms). Thus, whiskey enhanced vulnerability to atrial fibrillation and flutter in patients without heart failure or cardiomyopathy, substantiating the "holiday heart" syndrome.

Cardiomyopathy in the setting of chronic ethanol ingestion is associated with conduction disturbances and arrhythmias (1–6), especially atrial fibrillation and flutter. The "holiday heart" syndrome is the onset of atrial tachyarrhythmias after a bout of alcohol abuse, in the absence of overt cardiomyopathy or congestive heart failure (6). This implies a direct arrhythmogenic effect of ethanol that should be avoided by those otherwise at risk for atrial fibrillation or flutter, such as patients with valvular heart disease. The evidence for a holiday heart syndrome is mainly an increased incidence of paroxysmal atrial fibrillation and flutter on weekends or winter holidays. We investigated the arrhythmogenic effect of ethanol by examining vulnerability to atrial fibrillation and flutter with the extrastimulus technique after acute whiskey ingestion.

Methods

Study patients. Fourteen men, aged 43 to 75 years, underwent right atrial extrastimulation after informed written consent, to study the effects of whiskey. They were studied in the postabsorptive state at least five half-lives after cardioactive drugs were discontinued. Eleven patients were habitual alcohol abusers, and seven of these patients were studied before and after whiskey. Four of the 11 had experienced atrial fibrillation or flutter when drinking alcohol, but were not given whiskey because initial extrastimulation resulted in atrial vulnerability or sustained atrial fibrillation or flutter (discussed later). Five of the seven who were given whiskey had presented with a paroxysm of atrial fibrillation or flutter after a bout of ethanol ingestion that was typical of the holiday heart syndrome, without overt heart failure, while the other two patients gave only a history of palpitation. The remaining three patients were not alcohol drinkers and were studied for syncope and sinus bradycardia. They had neither palpitation nor recorded paroxysms of atrial fibrillation or flutter. Their sinus node recovery times were 1,300, 1,500 and 1,640 ms, respectively.

None of the 14 patients studied manifested heart failure, valvular disease or hypertension. One patient had electrocardiographic evidence of left ventricular hypertrophy and another had radiographic evidence of cardiomegaly. None had electrocardiograms indicating left atrial enlargement. Echocardiograms excluded mitral stenosis and did not suggest ventricular enlargement, but two patients had mild left atrial enlargement. None of the alcohol abusers presenting clinically with atrial fibrillation or flutter manifested congestive heart failure during the arrhythmia.

Protocol. Pulmonary capillary wedge pressures were monitored in the seven alcohol abusers who were later given whiskey. Right atrial stimulation was performed with use of the two distal poles of a quadripolar catheter, fluoroscopically positioned at the high lateral right atrium. Recordings were made from the proximal pair with filter settings of 30 to 500 Hz, using an Electronics for Medicine VR-16 oscilloscopic recorder. Single rectangular 2 ms twice-threshold extrastimuli were applied in 10 ms decrements after eight beats of atrial drive at 500 ms cycle lengths (120 beats per min) until atrial vulnerability or refractoriness was observed.

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Vulnerability to atrial fibrillation or flutter was diagnosed by more than 1 second of disorganized atrial activity or more than three depolarizations with cycle lengths of less than 250 ms, recorded from the atrial leads after the applied extra beat (7–9). If sustained atrial fibrillation did not result, stimulation was repeated at two additional right atrial sites.

Atrial vulnerability was not recorded in 10 of the 14 patients, and these 10 drank either 60 ml (2 patients) or 120 ml (8 patients) of 86 proof whiskey (43% ethanol) in 30 minutes. Extrastimulation was repeated 30 minutes after the drink. Blood was then drawn for determination of ethanol levels within 1 hour of the drink. In nine patients, extrastimulation was done at two additional right atrial sites before and after the whiskey.

Atrial functional refractory period was defined as the shortest interval achieved between the last driven beat and the extra beat. Dispersion of refractoriness was the difference between the longest and shortest refractory period.

Statistical comparisons were made with a paired *t* test.

Results

Induction of atrial fibrillation or flutter. Vulnerability to atrial fibrillation or flutter was demonstrated in two of the four alcohol abusers in the basal state, and two had sustained fibrillation or flutter. Therefore, these patients were not given whiskey to drink. Five of the 10 patients not vulnerable in the basal state had atrial fibrillation or flutter induced after whiskey (Fig. 1): two of five with holiday heart syndrome had atrial fibrillation induced only after whiskey; an alcohol abuser with palpitation and two nondrinkers had atrial flutter induced after whiskey.

Ethanol levels. These averaged (± standard deviation) 72 ± 8 mg/100 ml (range 49 to 101)—71 mg/100 ml in vulnerable patients and 77 mg/100 ml in those not vulnerable. Ethanol levels were only 82 and 83 mg/100 ml in the two in whom holiday heart syndrome was replicated, and 50 and 68 mg/100 ml when atrial flutter was induced in the nondrinkers. Pulmonary capillary wedge pressure was normal in all seven drinkers before whiskey (6 to 13 mm Hg) and did not change after whiskey.

Atrial refractoriness. Whiskey did not significantly alter atrial refractoriness (Fig. 2). High right atrial functional refractory period averaged 297 ± 14 ms (standard error of the mean) before and 290 ± 12 ms after whiskey (probability [p] = not significant [NS]). However, refractory periods were shortened by more than 10 ms in four of five vulnerable patients (average 20 ms); the refractory periods were not shortened in those who were not vulnerable. Dispersion of functional refractory period was not widened by whiskey (57 ± 13 ms before and 47 ± 12 ms after consumption, p = NS). Dispersion widened in only three patients and, in fact, was narrowed by whiskey in four of the five patients vulnerable to atrial fibrillation or flutter. In these latter four vulnerable patients, vulnerability was seen at the sites with the shortest refractory periods.

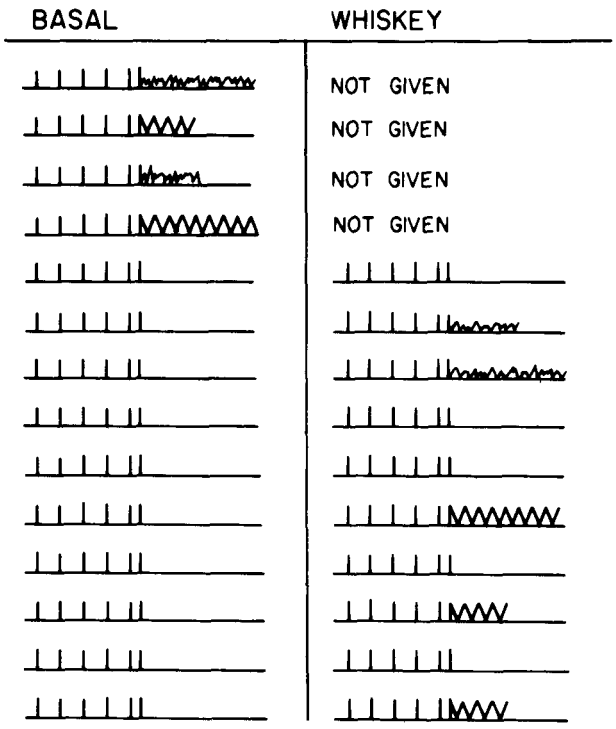

Figure 1. The results of atrial extrastimulation (basic stimulus cycle length of 500 ms) **(vertical lines)** in 14 patients. In the basal state, atrial fibrillation **(wiggly lines)** or flutter **(jagged lines)** was induced by extrastimuli **(vertical lines with short coupling)** in four alcohol abusers and was sustained in two. These patients were not given a drink. In the remaining 10 patients arrhythmias were not induced, and these patients were given whiskey. After whiskey, 5 of these 10 had atrial fibrillation or flutter induced, which was sustained in 2. The first 11 patients depicted had "holiday heart" syndrome, or at least palpitation when drinking. The last patients were not alcohol abusers and had sinus bradycardia

Discussion

We used whiskey to facilitate induction of atrial fibrillation and flutter by extrastimulation in 10 patients free of manifest heart failure. In three of seven alcohol abusers, the holiday heart syndrome was replicated. Failure to induce the syndrome in the remaining four could reflect the use of nonintoxicating doses. Two of three patients with sinus bradycardia had atrial flutter induced only after whiskey.

Mechanism of the atrial arrhythmias. Arrhythmias are frequent in patients with alcoholic cardiomyopathy (3,4), but are presumably caused by structural damage. Our patients did not have clinical evidence of cardiomyopathy, valvular heart disease or congestive heart failure, and left atrial size was generally normal. We cannot exclude preclinical myocardial dysfunction in our alcohol abusers, but whiskey caused no clinical or hemodynamic change, and a direct arrhythmogenic effect of the ethanol is suggested.

Arrhythmias from ethanol might be attributed to focal intramyocardial (10) or adrenal (11) release of catecholamines, nonuniform autonomic nervous system discharge or electrophysiologic effects of the metabolite acetaldehyde (10). Acetaldehyde was unlikely to have affected our pa-

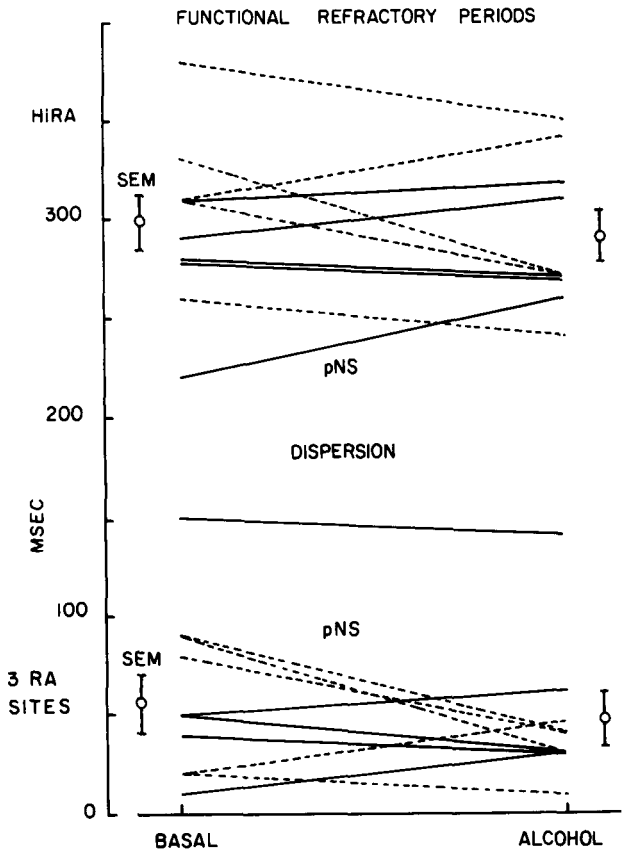

Figure 2. High right atrial (HiRA) functional refractory periods **(upper lines)** and dispersion of functional refractory periods **(lower lines)** at three disparate right atrial (RA) sites. All were measured at a 500 ms drive cycle length. The five patients with atrial vulnerability after whiskey are depicted by the **dashed lines.** High right atrial functional refractory periods averaged 297 ± 44 ms (mean ± standard error of the mean) before and 290 ± 12 ms after whiskey. Whiskey did not widen dispersion, which averaged 57 ± 13 ms before and 47 ± 12 ms after the drink. Induction of atrial fibrillation or flutter was generally associated with abbreviated refractoriness after whiskey, but with less dispersion of refractoriness

tients because extrastimulation was performed shortly after whiskey ingestion.

Role of atrial refractoriness. Dispersion of atrial refractoriness was not widened by ethanol. Gould et al. (12) and we found no abbreviation of refractory periods. We found that vulnerability to atrial fibrillation or flutter seemed to be associated with abbreviated refractoriness, but not at the vulnerable sites with the shortest refractory periods. Thus, dispersion of refractoriness actually narrowed in most vulnerable patients. Although this suggests that vulnerability to atrial fibrillation and flutter in the setting of ethanol is not caused by nonuniform changes in refractoriness, insufficient atrial sites might have been tested. Alternatively, focal conduction abnormalities caused by ethanol could have facilitated reentry. We did not measure conduction, but Gould and co-workers (12) found that ethanol prolonged intraatrial conduction.

In summary, a drink of whiskey facilitated the demonstration of vulnerability to atrial fibrillation and flutter in some alcohol abusers free of manifest cardiomyopathy and in some nondrinkers with bradycardia. Holiday heart syndrome was replicated by a small dose of whiskey. The mechanism was not delineated, but these observations suggest that perhaps social doses of ethanol can be harmful to those at risk for atrial fibrillation or flutter, as in the presence of sinus node or valvular disease.

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